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Review Article

Modern approaches to the treatment of atopic dermatitis in children: The role of biologics and Janus kinase inhibitors

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ABSTRACT

Atopic dermatitis (AD) is a chronic, inflammatory skin disease that often begins in early childhood and significantly affects the quality of life of patients and their families. Conventional treatments include emollients, corticosteroids, and calcineurin inhibitors; however, these are often insufficient in moderate-to-severe cases. In recent years, novel targeted therapies, including biologics and Janus kinase (JAK) inhibitors, have shown promising clinical results in pediatric patients. Dupilumab and tralokinumab, monoclonal antibodies, have demonstrated significant improvement in AD symptoms, including reduction of pruritus and inflammation. JAK inhibitors, such as upadacitinib, abrocitinib, and baricitinib, modulate cytokine pathways involved in AD pathogenesis, leading to rapid and sustained improvement of skin lesions. These novel therapies exhibit favorable safety profiles and enable personalized approaches. This article reviews current evidence on the efficacy and safety of dupilumab, tralokinumab, and JAK inhibitors in the pediatric population, highlighting their role in modern AD management.

Key words: Atopic dermatitis, Children, Treatment, Janus kinase inhibitors, Biological therapy

INTRODUCTION

Atopic dermatitis (AD) is the most common chronic inflammatory skin disease. It affects 15–20% of children and 3–10% of adults worldwide [1-3]. In children, it usually manifests before the age of 5, but it can develop at any age [4]. Approximately 75% of individuals with childhood-onset AD experience spontaneous remission of symptoms before reaching adolescence. In some patients whose symptoms do not resolve in childhood, AD symptoms persist with varying degrees of severity into adulthood. In some individuals, symptoms are constant, while in others, the disease is chronic with periods of relapse [4]. Atopic dermatitis, especially moderate to severe, is characterized by the presence of inflammatory skin lesions and intense pruritus, which results in reduced quality of life (QoL) in both patients and their caregivers [1,2,4,5]. This disease is a significant public health problem in developing countries, as its prevalence is high and the incidence is increasing in most countries [2]. Numerous guidelines for the management of atopic dermatitis in children have been published in recent years [6]. The disease usually requires ongoing treatment. Maintaining a therapeutic response and a manageable safety profile are important goals of long-term treatment for atopic

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dermatitis. In clinical practice, topical therapies are the mainstay of treatment. Systemic therapies are added in cases of more severe or refractory disease [7]. Individualized treatment is necessary, as not all patients respond equally to the same medications. Newly introduced therapies, as well as those under investigation, offer hope for patients with atopic dermatitis [8]. This review will discuss available drug therapies for atopic dermatitis in children.

MATERIAL AND METHODS

This study is a narrative review based on an analysis of current literature regarding modern therapeutic approaches for the treatment of atopic dermatitis (AD) in children using biologic agents and Janus kinase (JAK) inhibitors. A literature search was conducted using the PubMed, Scopus, and Web of Science databases. Publications evaluating the efficacy and safety of dupilumab, tralokinumab, and the JAK inhibitors upadacitinib, abrocitinib, and baricitinib in the pediatric population were included in the analysis. Randomized clinical trials, observational studies, meta-analyses, and review articles published in recent years were considered. The analyzed outcomes included treatment efficacy, effects on the severity of skin lesions, reduction of pruritus, and the safety profiles of the investigated therapies. The collected data were synthesized and qualitatively analyzed to assess the role of these novel therapies in the management of moderate-to-severe atopic dermatitis in children.

RESULTS

Pathogenesis of Atopic Dermatitis as a Basis for Targeted Therapy

The pathogenesis of AD is complex and involves a complicated interaction between epidermal barrier dysfunction, excessive activation of the immune system and microbiological dysbiosis [9].

In atopic dermatitis, loss-of-function mutations in filaggrin are considered one of the most important genetic factors predisposing to the development of the disease. These mutations correlate with early onset and a more severe course of the disease [4,10]. Furthermore, specific proteases, their inhibitors, and cytokines are involved in maintaining normal skin structure [11]. The type 2 inflammatory response, a dominant feature of atopic dermatitis, leads to increased production of cytokines, which induce epidermal barrier dysfunction. In particular, secondary release of the cytokines IL-4, IL-13, IL-5, and IL-31 has been demonstrated, which in turn activate other immune cells (e.g., macrophages and B lymphocytes), leading to the initiation of inflammatory cascades involved in the pathogenesis of the disease [9,12]. It has also been shown that the JAK–STAT signaling pathway is crucial in the pathogenesis and further course of atopic dermatitis [11].

The increasingly detailed understanding of the molecular and immunological pathways involved in the pathogenesis of atopic dermatitis is directing the attention of researchers and clinical practitioners to modern targeted therapies, including Janus kinase (JAK) inhibitors such as abrocitinib, baricitinib, and upadacitinib, as well as biologics, including dupilumab and tralokinumab, which selectively modulate type 2 inflammation.

Medicines

Dupilumab

Dupilumab is a monoclonal antibody against human immunoglobulin G4. The mechanism of action of dupilumab is to block signalingIL-4/IL-13 [13]. Cytokines IL-4 and IL-13 play a key role in the pathogenesis of allergic diseases and asthma, conditioning the humoral immune response dependent on T lymphocytes. IL-4 signals through the type I receptor (specific for IL-4) and the type II receptor, common to IL-4 and IL-13. Activation of the receptors leads to the phosphorylation of Janus family kinases (JAK1, JAK3, TYK2) and the activation of STAT signaling pathways, mainly STAT6 and STAT3. Dupilumab, by binding IL-4, blocks IL-4 and IL-13 signaling, leading to the inhibition of the inflammatory and immune response, hby inhibiting receptor signaling through the JAK-STAT pathway. This pathway plays a significant role in the main pathological mechanisms of atopic dermatitis, including impaired skin barrier function, antibody class switching to IgE, and TH2 cell differentiation [14,15]. Systemic blocking of the IL-4Rα subunit leads to a reduction in the number of circulating memory B cell type 2 (MBC2) cells and total IgE levels in pediatric patients with atopic dermatitis [16].

Dupilumab is approved for the treatment of moderate to severe atopic dermatitis. Female gender, young age, absence of allergic rhinitis, low body mass index (BMI), and low blood eosinophil counts are factors associated with a favorable response to dupilumab [17].

A systematic review and meta-analysis of randomized, placebo-controlled trials assessing the efficacy and safety of dupilumab in atopic dermatitis was conducted. The analysis of randomized trials confirmed the high efficacy and safety of dupilumab in patients with atopic dermatitis. Analysis of 14 studies including adults and children and adolescents demonstrated a significant reduction in the severity of skin lesions (EASI, SCORAD) and pruritus compared with placebo. Treatment with dupilumab significantly increased the percentage of patients achieving EASI 75 and improvement on the IGA scale. Clinical benefits also translated into a significant improvement in quality of life. The safety profile was comparable to placebo, with no increase in the incidence of adverse events. Efficacy and safety were consistent in both adults and children and adolescents [18].

A retrospective cohort study assessed whether dupilumab could inhibit the progression of the atopic march in children with atopic dermatitis. Analysis of data from over 4,000 patients showed that children treated with dupilumab were less likely to develop asthma or allergic rhinitis than those receiving conventional immunomodulators. The three-year risk of progression of the atopic march was significantly lower in the dupilumab group. This treatment was also associated with a significant reduction in the risk of developing asthma and allergic rhinitis. The greatest benefit was observed in younger children, particularly in asthma prevention. These results suggest that dupilumab may not only control atopic dermatitis but also limit the long-term progression of atopic diseases [19].

A 16-week, randomized, double-blind, phase III study evaluated the efficacy and safety of dupilumab in children aged 6 months to less than 6 years with moderate to severe atopic dermatitis inadequately controlled by topical therapy. Dupilumab administered every 4 weeks in combination with topical corticosteroids significantly outperformed placebo after 16 weeks of treatment. A significantly higher proportion of children achieved clear or almost clear skin (IGA 0–1) and improved EASI-75. Treatment was associated with significant clinical improvement even within a short period of follow-up. The frequency of adverse events was similar in both groups, and most were mild. Conjunctivitis occurred more frequently with dupilumab, but it was not severe and did not lead to treatment discontinuation. The results confirm that dupilumab is an effective and well-tolerated treatment option even in the youngest children with more severe atopic dermatitis [20].

Upadacitinib

Upadacitinib is a selective inhibitor of Janus kinases (JAKs) that acts by competing with adenosine triphosphate (ATP), leading to inhibition of kinase activity and phosphorylation of downstream effector proteins. In cellular studies, it demonstrates greater selectivity for JAK1 compared to JAK2, JAK3, and TYK2. Selective JAK1 inhibition enables inhibition of key cytokines and biological functions involved in the pathogenesis of immune-mediated inflammatory diseases, while relatively preserving signaling of other JAK-dependent cytokines. In vivo studies have shown that upadacitinib reversibly and concentration-dependently inhibits IL-6 and IL-7 cytokine signaling, as confirmed by assessing IL-6-induced STAT3 phosphorylation and IL-7-induced STAT5 phosphorylation in ex vivo studies [21].

Upadacitinib is an oral, selective and reversible JAK inhibitor approved for the treatment of multiple immune-mediated inflammatory diseases including atopic dermatitis (AD) [22]. Data from three randomized, double-blind, placebo-controlled phase III clinical trials conducted in more than 20 countries evaluating the long-term efficacy and safety of upadacitinib in adolescents with moderate-to-severe atopic dermatitis were analyzed. The studies enrolled 542 patients aged 12–17 years and were followed up to 76 weeks of treatment. A total of 542 adolescents participated in the study. Upadacitinib was administered at doses of 15 mg or 30 mg, either alone or in combination with topical corticosteroids. After 76 weeks, most patients maintained or improved their clinical response. Approximately 85–90% of patients treated with the 15 mg dose and 83–96% of those treated with the 30 mg dose achieved EASI-75. Improvement in the vIGA-AD scale (0/1) and reduction in pruritus (WP-NRS $\geq$4) were also maintained over time. Among adolescents with a HADS-A or HADS-D score of 8 or higher at baseline (approximately half of the study population), the proportion of patients free of anxiety and depression was also numerically higher with upadacitinib than with placebo across all studies. Sleep improved compared with placebo for both upadacitinib doses. The most common adverse events reported in adolescents receiving upadacitinib were acne, headache, upper respiratory tract infection, elevated creatine kinase (CPK), and nasopharyngitis [23-25].

A meta-analysis of randomized, controlled trials in patients with moderate to severe atopic dermatitis, including both adults and adolescents aged ≥12 years, showed that upadacitinib was significantly superior to placebo in the treatment of moderate to severe atopic dermatitis. A significantly higher proportion of patients achieved an EASI 75 (RR = 3.86) and achieved complete or almost complete clearance of skin lesions, as assessed by an EASI 100 (RR = 13.09). Treatment was also associated with a significant reduction in pruritus, as measured by the WP-NRS (RR = 4.44). Clinical improvement was also confirmed by the investigator's global assessment (vIGA) (RR = 5.96). Acne, upper respiratory tract infection, and nasopharyngitis were commonly reported adverse events and occurred more frequently in patients receiving upadacitinib than in those receiving placebo. The analysis revealed no significant differences in efficacy between the 15 mg and 30 mg doses. The results confirm the high efficacy of upadacitinib with an acceptable safety profile [26].

We also examined the impact of once-daily upadacitinib on symptoms and quality of life over 16 weeks in adults and adolescents with moderate-to-severe atopic dermatitis in the Measure Up 1 and 2 clinical trials. The analysis included 1,683 adults and adolescents receiving upadacitinib 15 mg, 30 mg, or placebo for 16 weeks. Significantly more patients treated with upadacitinib achieved a clinically meaningful reduction in pruritus (WP-NRS ≥4) as early as week 1 of treatment compared with placebo. This improvement continued through weeks 4–6 and was maintained through week 16, with 47.1% of patients on the 15 mg dose and 59.8% on the 30 mg dose. Beneficial effects were also observed in skin pain, sleep, daily functioning, emotional state, mental health, and quality of life. Patients also reported significant improvements in their perception of disease severity and treatment effectiveness. The results confirm that upadacitinib monotherapy provides rapid and sustained improvement in symptoms and quality of life compared to placebo [27].

An analysis of long-term treatment of inflammatory diseases, including AD, with upadacitinib was also performed, demonstrating good tolerability. The analysis included 8632 patients, including 2683 with AD, treated with upadacitinib for 27,164.2 patient-years [28].

Tralokinumab

Genes related to the skin barrier and inflammation in the skin of patients with atopic dermatitis correlate with IL-13 expression. Tralokinumab, a fully human monoclonal IgG4 antibody, specifically binds to IL-13 with high affinity, preventing interaction with the IL-13 receptor and subsequent signaling, thereby inhibiting the induction of inflammatory mediators viainhibition of the expression of relevant genes in keratinocytes or skin fibroblasts [29]. Tralokinumab has been approved for the treatment of moderate to severe atopic dermatitis in adult patients eligible for systemic treatment [30].

The 52-week phase III ECZTRA 6 study, which enrolled 289 patients aged 12 to 17 years, evaluated the efficacy and safety of tralokinumab monotherapy in adolescents with moderate-to-severe atopic dermatitis. Patients were aged 12 to 17 years. After 16 weeks, significantly more patients treated with tralokinumab (150 mg and 300 mg) achieved an IGA 0/1 and EASI 75 response compared with placebo. Treatment was also associated with greater reductions in pruritus, SCORAD scores, and quality of life. In more than half of the patients who responded at week 16, efficacy was maintained through week 52 without the need for rescue treatment. Clinical responses (IGA 0/1 and EASI 75) were maintained in a significant proportion of patients during the open-label phase. Tralokinumab was well tolerated and was not associated with an increased risk of conjunctivitis [31].

The safety of long-term tralokinumab was assessed in seven placebo-controlled studies and the ECZTEND study. A total of 2,693 patients received tralokinumab for up to 4.5 years, allowing for the assessment of long-term exposure in adults and adolescents with moderate to severe AD. The safety profile was comparable to placebo. Most adverse events were mild or moderate, most commonly nasopharyngitis, conjunctivitis, and injection site reactions. However, exacerbations of AD, asthma, and severe skin infections were observed less frequently [32].

A clinical trial was conducted to evaluate the tolerability of an autoinjector with the recommended dose for most patients (300 mg tralokinumab) administered as a single injection every 2 weeks. The prefilled pen formulation was effective, well tolerated, and easy to use. The tralokinumab prefilled pen may offer a more convenient method of administering tralokinumab, with fewer injections per dose [33].

Studies evaluating the efficacy and safety of tralokinumab in children aged 6 to <12 years with

moderate to severe AD are currently underway (NCT05388760, NCT06311682).

Abrocitinib

Abrocitinib is a Janus kinase 1 (JAK1) inhibitor under investigation for the treatment of atopic dermatitis. Selective inhibition of JAK1 modulates the signaling of IL-4, IL-13, and other cytokines (e.g., IL-31, IL-22, and thymic stromal lymphopoietin) involved in disease pathogenesis [34]. Abrocitinib demonstrates high efficacy and a favorable safety profile in the treatment of moderate to severe atopic dermatitis (AD) in adolescents and adults. A meta-analysis of 12 randomized trials involving 5644 participants showed a significantly higher percentage of patients achieving improvement on the IGA, EASI, PP-NRS, and POEM scales with abrocitinib compared with placebo. Greater efficacy was observed with the 200 mg dose compared with the 100 mg dose. Adverse events were rare and comparable to placebo, and were mostly mild or moderate. The results confirm that abrocitinib is an effective and safe option for the long-term treatment of atopic dermatitis [35]. Adverse events were reported more frequently in the active treatment groups, but serious events were rare and comparable to placebo. There were no treatment-related deaths. The results confirm that oral abrocitinib is an effective and well-tolerated treatment option in adolescents and adults with moderate-to-severe atopic dermatitis [36].

An integrated analysis of data from the JADE studies assessed the long-term efficacy and safety of abrocitinib in adolescents with moderate-to-severe atopic dermatitis. Treatment efficacy was maintained up to week 112, and safety was assessed with exposure up to 4.6 years. At week 112, a high and comparable proportion of patients treated with the 100 mg and 200 mg doses achieved clinical response, including EASI-75 (approximately 83–85%), EASI-90 (60–62%), and IGA 0/1 (57%). The results confirm durability of clinical response regardless of dose. The safety profile was consistent with previous observations, with a slightly higher adverse event rate for the 200 mg dose, but without new safety signals. Serious adverse events and treatment discontinuations were rare, and the confidence intervals between doses overlapped. The overall data support the long-term use of abrocitinib in adolescents with atopic dermatitis [37]. The long-term safety analysis of abrocitinib showed an overall favorable drug profile across all age groups. The lowest percentage of adverse events of particular significance was observed in adolescents [38]. Studies indicate that oral abrocitinib combined with topical therapy is significantly more effective than topical placebo in adolescents with moderate to severe disease [39].

Furthermore, the efficacy of abrocitinib was assessed in patients with moderate to severe atopic dermatitis with or without concomitant allergic diseases. More than half of the study participants had at least one allergic disease, most commonly asthma, allergic rhinitis, conjunctivitis, or food allergy. Regardless of the presence of comorbidities, treatment with abrocitinib (100 mg or 200 mg) led to significantly higher rates of IGA 0/1, EASI-75, reduction of pruritus (PP-NRS4), and improvement in quality of life (DLQI) compared with placebo. Improvements in POEM, SCORAD, and PSAAD scores were greater in the abrocitinib-treated groups than in the placebo group. The drug was generally well tolerated, with most adverse events being mild or moderate in severity [40].

Baricitinib

The JAK–STAT pathway plays a key role in immune function and hematopoiesis and is a key mechanism involved in the development of many immune-mediated diseases, such as atopic dermatitis [41]. Baricitinib is an oral, selective inhibitor of the Janus kinases JAK1 and JAK2, approved for the treatment of moderate to severe atopic dermatitis (AD) in adults [41-44]. Due to its inhibition of multiple JAK kinases, baricitinib is used to treat immune-mediated diseases other than atopic dermatitis and is a first-generation JAK inhibitor. This drug was recently approved by the European Medicines Agency (EMA) for the treatment of moderate to severe atopic dermatitis in adult patients who are candidates for systemic therapy [41]. Studies indicate that it may also be a potential therapeutic option for pediatric patients.

A randomized, double-blind, placebo-controlled phase III study (BREEZE-AD PEDS) evaluated the efficacy and safety of three doses of baricitinib in combination with low- or intermediate-strength topical corticosteroids in pediatric patients with moderate-to-severe atopic dermatitis. A total of 483 patients were enrolled, with a mean age of 12 years. Baricitinib at a dose equivalent to 4 mg demonstrated statistically significant superiority over placebo ($P < 0.05$) for all endpoints assessed at week 16. Baricitinib treatment was associated with significant improvements in sleep initiation and a reduced need for topical corticosteroids. The discontinuation rate due to adverse events was low, at 1.6% in the placebo group and 0.6% in the baricitinib

group. There were no deaths or serious adverse events such as thromboembolic events, cardiovascular events, cancers, gastrointestinal perforations or opportunistic infections [42].

After the primary endpoint at week 16, patients who achieved a complete or partial response to treatment continued long-term therapy with baricitinib 4 mg for up to 4 years. Treatment efficacy in these patients who continued double-blind treatment was generally maintained up to week 52 [43]. Some studies suggest that the risk of upper respiratory tract infections was increased with baricitinib 2 mg and 4 mg [45]. A study comparing the efficacy and safety of dupilumab and baricitinib showed that although baricitinib did not demonstrate equivalent efficacy to dupilumab, higher doses (2 mg and 4 mg) demonstrated superior therapeutic effects compared with placebo. Therefore, it remains a viable treatment option [45].

DISCUSSION

Atopic dermatitis therapy aims to achieve the longest possible periods of remission by strengthening the epidermal barrier, reducing inflammation and pruritus, and eliminating factors that trigger flare-ups [46,47]. Treatment of atopic dermatitis has advanced significantly in recent years [48]. Both Polish and international recommendations—including those of the AAD, the European Task Force on Atopic Dermatitis (ETFAD)/EADV, and EuroGuiDerm—emphasize the need for individualized treatment depending on the patient's age and clinical presentation [46,47]. Current atopic dermatitis therapy includes routine skin care, avoidance of trigger factors, and phototherapy, both topical and systemic. According to current guidelines, the treatment of atopic dermatitis depends primarily on the severity of the disease [48,49]. In patients with moderate to severe atopic dermatitis, in whom proper skin care, topical treatment, and UV light therapy do not provide adequate symptom control, systemic therapy is considered. Systemic therapy may also enable a reduction in the dose of topical corticosteroids (GCs) in patients who require long-term use of high doses of potent GCs to control their symptoms. Systemic treatment includes immunosuppressive drugs, biological therapies, and Janus kinase (JAK) inhibitors. Before the introduction of molecularly targeted therapy, traditional immunosuppressive drugs such as cyclosporine, methotrexate, mycophenolate mofetil, and azathioprine were the mainstays of therapy. Most of these drugs were used off-label [49].

Current trends in research and clinical practice focus on the increasing importance of biologics and JAK kinase inhibitors. New Janus kinase inhibitors – baricitinib, abrocitinib, and upadacitinib – are classified as rapid-acting drugs, whereas Th2-blocking drugs (IL4/IL13 or IL13 inhibitors) (dupilumab and tralokinumab, respectively) require several weeks to achieve full efficacy [49]. The results of a network meta-analysis show that these JAK inhibitors demonstrate significantly greater efficacy than placebo in the treatment of moderate to severe atopic dermatitis. Furthermore, an analysis of available studies indicates upadacitinib as the most effective drug among systemic therapies in the treatment of atopic dermatitis in children. This drug achieves the best results compared to other treatment regimens [11,50]. At the same time, the need for further actions aimed at minimizing the risk of therapy-related adverse events is emphasized [11], (Table 1).

IL-4/IL-13 (dupilumab) and IL-13 (tralokinumab) inhibitors are also an effective and safe treatment method even in very young children with advanced AD [20,31].Studies show that treatment with both tralokinumab and dupilumab results in significant reduction of pruritus, improvement in SCORAD scores, and quality of life [18,31]. Furthermore, dupilumab has been shown to contribute to limiting the long-term progression of atopic diseases, in addition to controlling the symptoms of atopic dermatitis [19]. Dupilumab therapy was associated with a slightly increased risk of conjunctivitis, but this was mild and did not lead to treatment discontinuation. Tralokinumab, on the other hand, was well tolerated, with no increased incidence of any adverse events reported [20,31].

Eleven studies involving 2,352 pediatric patients (<18 years of age) were analyzed to assess the differences in efficacy and safety between dupilumab and newly approved biologics and JAK inhibitors in the treatment of pediatric atopic dermatitis. Efficacy was assessed using the Investigator's Global Assessment (IGA), the Itch Numeric Rating Scale (NRS), and the Eczema Area and Severity Index (EASI). The investigators concluded that upadacitinib (30 mg) was most effective in achieving EASI-75, EASI-90, and IGA 0/1. Dupilumab, on the other hand, demonstrated high efficacy and a more favorable safety profile than JAK inhibitors. Tralokinumab provided the best reduction

Table 1: Comparison of drugs used in the treatment of atopic dermatitis in children

Bow	Dupilumab	Abrocitinib	Tralokinumab	Baricitinib	Upadacitinib
Registration age in Poland	From 6 months	From 12 years of age	From 12 years of age	No registration for children	From 12 years of age
EU	From 6 months	From 12 years of age	From 12 years of age	No registration for children	From 12 years of age
Form of the drug	Subcutaneous injections	Oral	Subcutaneous injections	Oral	Oral
Dosage schedule	Depends on age and weight	1x 24 hours	1x 2 weeks	1x 24 hours	1x 24 hours
Mechanism of action	IL-4 / IL-13 signal blockade	Selective JAK1 inhibitor	Antibody against IL-13	JAK1/JAK2 inhibitor	Selective JAK1 inhibitor
Speed of action	Improvement in EASI in 4 weeks of treatment	Very fast, EASI improvement from 1 week of treatment	The slowest onset of action, first effects after 4-8 weeks	EASI improvement after just 1-2 weeks of treatment	The fastest, EASI improvement already in the 1st week of treatment
Antipruritic effect	Reduction after just 2 weeks of treatment	More effective than dupilumab	Reduction of itching in just 1 week of treatment	The weakest itching reduction	Fastest itching reduction, after 2-7 days improvement by ≥ 4 points on the scale NRS of pruritus severity at week 16 compared to placebo
Most common side effects	Injection site reactions, conjunctivitis, joint pain, oral herpes and eosinophilia	Nausea, headache, acne, herpes simplex, increased blood creatine phosphokinase activity, vomiting	Upper respiratory tract infections, injection site reactions, inflammation conjunctiva	Increased LDL cholesterol, upper respiratory tract infections, nausea, viral infections chickenpox and shingles	Upper respiratory tract infection, acne, herpes simplex, headache, increased phosphokinase activity creatine (CPK) in the blood, cough, folliculitis, abdominal pain, nausea, neutropenia, fever and flu

in pruritus but was less effective in reducing skin symptoms. Baricitinib was considered the least effective agent. It was noted that JAK inhibitors produced a strong clinical response but were more often associated with adverse events [45]. The data we currently have largely concern adolescents, therefore further studies by age group in younger children and long-term assessment of the treatment safety are necessary [50].

CONCLUSIONS

Recent progress in the treatment of pediatric atopic dermatitis has been driven by the introduction of targeted therapies, including biologics and JAK kinase inhibitors, which enable more effective control of moderate to severe disease. JAK inhibitors, such as baricitinib, abrocitinib, and upadacitinib, represent a promising therapeutic option, particularly in children and adolescents with an inadequate response to topical therapy. Dupilumab and tralokinumab, monoclonal antibodies targeting key Th2 cytokines, also demonstrate favorable efficacy and safety profiles in the long-term treatment of pediatric patients with atopic dermatitis. Studies indicate that the use of modern systemic therapies contributes to a significant reduction in symptom severity and improved quality of life. To optimize the treatment of pediatric atopic dermatitis, individualized treatment selection is crucial, taking into account the patient's age, disease severity, and the safety profile of individual medications.

Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

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